Is Noninvasive Determination of Pulmonary Artery Pressure Feasible Using Deceleration Phase Doppler Flow Velocity Characteristics in Mechanically Ventilated Children With Congenital Heart Disease?

Arie P.J. van Dijk, MSc, Jeroen C.W. Hopman, MSc, John H.G.M. Klaessens, MSc, Tjeerd van der Werf, MD, and Otto Daniëls, MD

Noninvasive determination of pulmonary hemodynamics is important for the management of congenital heart disease complicated by pulmonary hypertension. Flow deceleration is less influenced by right ventricular function and would allow more accurate estimation of pulmonary hemodynamics than acceleration. Respiratory influences on pulmonary blood flow are exaggerated by mechanical ventilation. Doppler-derived pulmonary artery (PA) blood flow velocity characteristics were therefore compared with pulmonary hemodynamic parameters in 42 mechanically ventilated children, aged 0.2 to 14.8 years (mean ± SD 6.7 ± 4.9). Mean PA pressure ranged from 11 to 47 mm Hg (21 ± 9 mm Hg). Pulmonary hypertension was present in 14 patients. Significant differences were found between patients with and without pulmonary hypertension in maximal velocity (1.03 ± 0.22 vs 0.88 ± 0.18 m/s), acceleration time (119 ± 39 vs 136 ± 29 ms), maximal acceleration (17.6 ± 6.4 vs 13.1 ± 4.0 m/s^2), mean acceleration (9.3 ± 2.6 vs 6.7 ± 2.0 m/s^2), and mean deceleration (4.5 ± 1.0 vs 3.8 ± 0.8 m/s^2). In contrast to our hypothesis of the deceleration phase–derived parameters, only maximal deceleration correlated with PA pressure. Acceleration parameters showed closer relations with PA pressures, but correlations were generally low and did not permit accurate prediction of PA pressure (SEE 5 to 11 mm Hg), PA resistance (SEE 1.14 $U \cdot m^2$) or PA driving force (SEE 7 mm Hg). An analysis that took respiratory phase into account did not improve correlations. Measurement of mean acceleration, maximal deceleration, and rate-corrected preejection period permitted for accurate discrimination between the presence or absence of pulmonary hypertension, with positive and negative predictive values being 92% and 90%.

In mechanically ventilated children with congenital heart disease, accurate noninvasive PA pressure assessment is not possible. Accurate predictions for the presence of pulmonary hypertension can be made by measurement of both acceleration and deceleration parameters.

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Many investigations have already been published concerning the noninvasive determination of pulmonary artery (PA) pressure and PA vascular resistance using changes in systolic blood flow velocity wave in the right ventricular outflow tract[1–4] or main PA.[5–12]

The acceleration phase–derived Doppler parameters have been thoroughly investigated with conflicting results. In pulmonary hypertension, resistance to flow is not only changed during acceleration, but also during deceleration. Moreover, clear changes have been described in the deceleration part in pulmonary hypertensive patients.[13] Because the deceleration phase of pulmonary blood flow is less influenced by right ventricular systolic function, deceleration phase–derived parameters would be more afterload dependent and may allow more accurate estimation of PA vascular resistance and PA pressure.

During intermittent positive pressure ventilation, the pulmonary blood flow velocity wave is influenced by changes in intrathoracic pressure and by respiratory-related changes of sample-volume position.[14,15]

The aim of this study is to determine the feasibility to predict PA pressure and resistance by using acceleration, but, especially, deceleration phase velocity characteristics in mechanically ventilated children with congenital heart disease.

METHODS

Patients: Forty-two pediatric patients, aged 0.2 to 14.8 years (mean ± SD 6.7 ± 4.9) were included in the study and underwent diagnostic cardiac catheterization for evaluation of congenital heart disease (Table I). Written informed consent by the parents was obtained in all patients. The study has been ap-

From the Children's Heart Centre and Department of Cardiology, University Hospital Nijmegen, Nijmegen, The Netherlands. This study was supported by Grant 88.220 from the Netherlands Heart Foundation, The Hague, the Netherlands. Manuscript received October 30, 1995; revised manuscript received and accepted June 24, 1996.

Address for reprints: Arie P.J. van Dijk, MD, Department of Cardiology, University Hospital Nijmegen, P.O. Box 9101, 6500 HB Nijmegen, The Netherlands.

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TABLE 1 Age, Pulmonary Hemodynamics, and Diagnosis of Participating Patients

Patient Number	Age (yr)	PA Pressure (mm Hg)			PA Resistance ($U \cdot m^2$)	Diagnosis
		Systolic	Mean	Diastolic		
1	4.4	22	13	9	0.43	VSD
2	1.4	19	15	11	0.47	AS, balloon valvuloplasty
3	13.9	62	37	21	—	ASD-II
4	5.1	39	29	21	0.96	VSD, corrected coarctation
5	11.4	19	15	11	1.58	Noonan's syndrome
6	10.5	25	21	17	1.21	AS, AR, balloon valvuloplasty
7	0.5	48	33	23	3.07	VSD
8	5.0	53	36	23	1.61	VSD
9	9.5	27	18	12	1.02	Corrected coarctation, AS
10	0.2	69	47	33	3.19	Pulmonary lymphangiectasia
11	3.9	24	18	11	1.10	ASD-sinus venosus type
12	3.5	22	17	11	0.76	Corrected coarctation
13	5.6	23	12	6	0.58	VSD
14	0.4	32	18	10	0.58	VSD, subvalvular AS
15	3.3	22	16	12	0.89	ASD-II
16	0.9	25	20	17	3.19	AS
17	1.0	43	30	22	5.24	ASD-I, muscular VSD
18	8.6	23	15	9	1.06	ASD-II, corrected coarctation
19	2.4	22	16	9	—	VSD
20	0.6	54	32	14	2.47	VSD, ASD-II, trisomy 21
21	8.7	27	17	9	3.85	VSD
22	13.1	27	14	6	0.43	ASD-II, mitral regurgitation
23	13.7	31	22	15	2.14	VSD
24	4.9	21	11	5	2.93	Corrected AVSD, mitral regurgitation
25	1.6	17	11	7	0.13	ASD-II, part. aberrant pulmonary venous return
26	13.0	23	15	11	1.24	VSD, coarctation
27	0.9	19	13	9	1.55	AS
28	2.3	69	44	24	2.98	AVSD, trisomy 21
29	3.3	25	15	10	0.40	AS, AR, balloon valvuloplasty
30	9.1	29	22	16	0.36	VSD
31	7.0	25	19	14	0.62	VSD
32	14.1	50	26	12	3.99	AS, AR, corrected PDA
33	11.4	24	19	14	2.09	ASD-I, mitral regurgitation
34	8.5	22	18	13	0.45	ASD-I, mitral regurgitation
35	13.4	29	19	15	1.17	Corrected AVSD, mitral valve replacement
36	4.1	17	14	11	1.18	Sweet syndrome, abnormal systemic arteries
37	3.3	19	14	12	0.60	Coarctation, bicuspid aortic valve
38	7.8	29	18	9	—	VSD
39	10.9	25	19	13	0.69	VSD
40	2.0	28	21	15	0.98	ASD-II
41	14.8	24	18	13	2.53	Corrected coarctation, balloon angioplasty
42	2.4	26	21	16	1.27	VSD

AR = aortic valve regurgitation; AS = aortic valve stenosis; ASD-I = atrial septal defect, primum type; ASD-II = atrial septal defect, secundum type; AVSD = complete atrioventricular septal defect; part. = partial; PDA = persistent ductus arteriosus; VSD = ventricular septal defect.

proved by the local committee on human experimental research. Excluded from the study were patients with transposition of the great arteries, patent ductus arteriosus, or right ventricular outflow obstruction (pressure difference >15 mm Hg). In those with left to right shunt, the ratio of pulmonary to systemic blood flow was 2.28 ± 1.15 (range 1.15 to 5.00). All patients were in sinus rhythm, 3 had complete right bundle branch block (QRS duration ≥0.12 second and rSr' in lead V_1).

Doppler echocardiographic measurements were obtained using a commercially available echo-Doppler apparatus (Toshiba SSH 65A, Toshiba Medical Systems, Tokyo, Japan) equipped with a 3.75-MHz transducer, with the possibility of performing combined 2-dimensional echocardiography and continuous-wave or single-gated pulsed-wave Doppler echocardiography. In the supine position, the pulmonary valve and PA were visualized from both short- and long-axis views using standard techniques from the second or third left parasternal space. To obtain the PA flow velocity wave, the sample volume (length 3 mm) was carefully placed in the middle of the PA, approximately 1 cm distal to the pulmonary valve, irrespective of the length of the PA using the appropriate pulse repetition frequency to avoid aliasing. Doppler frequency components <400 Hz were eliminated.

Diagnostic right-sided cardiac catheterization was performed in the catheterization laboratory during a steady hemodynamic state in mechanically ventilated patients (see later). All intracardiac pressures were measured with standard fluid-filled catheters connected to a strain-gauge manometer (Bentley Trantec pressure transducer model 800, volume displacement 0.04 mm^3/100 mm Hg, Irvine, California). Mean pulmonary capillary wedge pressure was determined with a Swan-Ganz catheter. Cardiac output

was obtained by thermodilution in triplicate.[16] The average was used in calculations. The pulmonary vascular driving force was computed by subtracting the mean pulmonary capillary wedge pressure from the mean PA pressure.

Oxygen saturations were derived from blood samples of all right-sided cardiac chambers, the PA, and the superior and inferior caval veins (Radiometer OSM-1 or OSM-2, Radiometer, Copenhagen, Denmark). Aortic and pulmonary venous oxygen saturations were obtained or were assumed to be 100%. Mixed venous oxygen saturation was calculated from the superior (SVC) and inferior (IVC) caval vein saturations: $(3 \cdot SVC + IVC)/4$. The ratio of pulmonary to systemic blood flow and pulmonary vascular resistance were calculated using oxygen saturations with standard equations.[17,18] The pulmonary blood flow was calculated by multiplication of this ratio with the average cardiac output.

Anesthesia: All children were catheterized under general anesthesia. All children aged >1 year were premedicated with midazolam 0.2 to 0.3 mg/kg. Induction of anesthesia was achieved with methohexital 25 mg/kg in children <20 kg and with etomidate 0.2 to 0.3 mg/kg, intravenously, in children ≥20 kg. Vecuronium 0.1 mg/kg, intravenously, was used for muscular paralysis. Continuation of anesthesia was achieved using halothane 0.5% to 1.5%, dinitric-oxide/oxygen mixture or propofol 0.05 to 0.25 mg/kg/min intravenously.

All children were mechanically ventilated using intermittent positive pressure (Siemens Servo 900 B/C, Erlangen, Germany) with tidal volumes of 8 to 10 ml/kg, mean peak pressures of 17.5 cm water and fractional inspired oxygen of 0.30 to 0.35. No positive end-expiratory pressure was used. The ventilatory frequency was adjusted to achieve normocapnic ventilation monitored by an end-expiratory partial pressure of carbon dioxide (range 4 to 6 kPa) (Hewlett-Packard Capnometer HP47210A with sensor model 14360, Waltham, Massachusetts).

Registration: PA pressures and PA blood flow velocity were simultaneously obtained. The Doppler velocity waves and lead II electrocardiogram were recorded on VHS videotape with registration speed equal to 75 mm/s for a 20-second period. The lead II electrocardiogram, the capnogram, and the PA pressure were digitized and stored using an on-line computer with a sample frequency of 200 or 300 Hz. The Doppler curves and the electrocardiogram were recorded on VHS videotape. To synchronize the Doppler curves with the PA pressure wave, the electrocardiogram, and the capnogram, a mark was recorded on both registrations simultaneously. In this way a beat-to-beat synchronization was achieved.

Onset of each QRS complex was determined from the lead II electrocardiographic signal using a specially designed QRS detection program. The starting points obtained were used for calculation of RR intervals and other time-referenced parameters.

The Doppler velocity curves of the main PA were obtained by manually tracing the outer borders of the Doppler velocity curve and digitized using an image-processing computer program. After visual examination of the pulmonary blood flow velocity curves (in all, >2,000 traces have been done), the curves were thought to be composed of 3 parts: the accelerating part of the curve, the top, and the decelerating part. A computer algorithm fitted the acceleration part and the top by a second-order polynomial function. In some curves, the deceleration part seemed to consist of 2 parts divided by a bending point, and was therefore fitted by a third-order polynomial function. From the obtained functions, maximal velocity, maximal acceleration, maximal deceleration, preejection period, acceleration time, deceleration time, and ejection time were computed. Mean acceleration and mean deceleration were calculated by division of maximal velocity by acceleration or deceleration time, respectively. To correct for heart rate dependency of time intervals, several parameters were divided by the RR interval or by the square root of the RR interval.

To investigate the effects of respiratory phase (i.e., the respiratory intrathoracic pressure changes on the measurements), a respiration curve was computed using the changes in the PA pressure signals due to respiration. From all recorded beats, an average pressure wave representing the average pressure change by cardiac contraction was calculated. Subtraction of the average curve from the pressure curve of each individual heart beat gives the pressure change due to respiration from which a respiration curve was constructed. Each beat was classified as being inspiratory, expiratory, end-expiratory, or end-inspiratory by determining the respiratory phase of its beginning and its ending. Beats having both a beginning and ending in inspiration or expiration were classified as inspiratory or expiratory, respectively. Beats beginning in expiration and ending in inspiration were classified as end-expiratory. Those starting during inspiration and ending during expiration were called end-inspiratory beats.

Because catheter movement artifacts were present, the pressure signals for each cardiac cycle were digitally filtered at 8 Hz, with 3 dB/octave by standard fast-Fourier transform procedures. The filtered pressure waves were used in further analysis. For each cardiac cycle, the systolic PA pressure, the diastolic PA pressure, and the mean PA pressure were obtained. Pulmonary hypertension was defined as mean PA pressure ≥20 mm Hg and/or systolic PA pressure ≥30 mm Hg.

For each measurement, the median of the first 6 consecutive cardiac cycles (irrespective of respiratory phase), was calculated and used in statistical analysis. In analyses concerning the respiratory phase, the values of the first beat of each phase were used. Data are presented as mean ± SD. To compare parameters between groups, the Wilcoxon 2-sample test or 1-way analysis of variance were used. Pearson correlation coefficients were calculated to determine correlations between Doppler parameters and PA pressure, the pulmonary vascular driving force, and

TABLE II Mean ± SD of Parameters in Children With (n = 14) and Without Pulmonary Hypertension (n = 28)

	All	Pulmonary Hypertension	No Pulmonary Hypertension
Age (yr)	6.7 ± 4.9	5.7 ± 5.9	7.2 ± 4.6
Heart rate (min^{-1})	88 ± 22	96 ± 24	84 ± 20
Length (cm)	115 ± 34	105 ± 39	120 ± 31
Weight (kg)	24 ± 15	20 ± 16	25 ± 14
PA systolic pressure (mm Hg)	30.5 ± 13.8	44.8 ± 15.5*	23.3 ± 3.7
PA mean pressure (mm Hg)	20.7 ± 8.6	30.2 ± 8.6*	16.0 ± 2.5
PA diastolic pressure (mm Hg)	13.2 ± 5.8	19.2 ± 5.5*	10.6 ± 2.7
Driving force (mm Hg)	9.2 ± 8.7	16.6 ± 11.7*	5.6 ± 3.0
PA resistance ($U \cdot m^2$)	1.56 ± 1.21	2.27 ± 1.40*	0.55 ± 0.95
Pulmonary to systemic flow ratio	1.88 ± 1.20	1.72 ± 1.12	2.20 ± 1.34
Maximal velocity (m/s)	0.93 ± 0.24	1.03 ± 0.22*	0.88 ± 0.18
Acceleration time (ms)	130.3 ± 33.2	119.4 ± 38.5*	135.8 ± 29.3
Deceleration time (ms)	230.6 ± 27.3	231.1 ± 29.4	230.3 ± 26.8
Maximal acceleration (m/s^2)	14.6 ± 5.3	17.6 ± 6.4*	13.1 ± 4.0
Maximal deceleration (m/s^2)	8.6 ± 2.2	9.5 ± 2.6	8.1 ± 1.8
Mean acceleration (m/s^2)	7.6 ± 2.5	9.3 ± 2.6*	6.7 ± 2.0
Mean deceleration (m/s^2)	4.0 ± 0.9	4.5 ± 1.0*	3.8 ± 0.8
Preejection time/ejection time	0.24 ± 0.07	0.22 ± 0.05	0.24 ± 0.07
Acceleration time/ejection time	0.36 ± 0.06	0.33 ± 0.07	0.37 ± 0.06
Acceleration time/RR interval	0.18 ± 0.04	0.18 ± 0.05	0.18 ± 0.03
Prejection period/$\sqrt{RR}$ interval ($ms^{-1/2}$)	3.16 ± 0.76	2.99 ± 0.61	3.24 ± 0.82
Acceleration time/$\sqrt{RR}$ interval ($ms^{-1/2}$)	4.86 ± 0.96	4.64 ± 1.19	4.97 ± 0.82
Deceleration time/$\sqrt{RR}$ interval ($ms^{-1/2}$)	8.72 ± 1.13	9.10 ± 1.06	8.53 ± 1.14

* $p < 0.05$ between pulmonary hypertension and no pulmonary hypertension.
PA = pulmonary artery.

TABLE III Classification of Patients Using Discriminant Function

	Using Discriminant Function Analysis		
	Doppler −	Doppler +	Total
Pressure −	27 (96%)	1 (4%)	28 (100%)
Pressure +	3 (21%)	11 (79%)	14 (100%)
Total	30 (71%)	12 (28%)	42 (100%)

pulmonary vascular resistance index. Multiple linear stepwise regression analysis was performed with Doppler parameters and respiration phase as independent variables to investigate the effects of the respiratory phase on pulmonary hemodynamics. To obtain regression equations, multiple stepwise regression was used with only Doppler parameters as independent variables. Multiple stepwise linear discriminant function analysis was performed to investigate which Doppler parameter best discriminated patients with and without pulmonary hypertension. A p value <0.05 was considered significant. Statistical analyses were performed using either SAS or SPSS software packages.

RESULTS

No significant differences in baseline patient characteristics were present (Table II). In pulmonary hypertension, maximal velocity, and maximal and mean acceleration were higher and acceleration time was significantly shorter. With regard to the deceleration parameters, only mean deceleration was significantly higher in pulmonary hypertensive patients. In those without and the 3 with right bundle branch block, maximal velocity (0.95 ± 0.19 vs 0.61 ± 0.15 m/s^2), maximal acceleration (15.0 ± 5.3 vs 9.3 ± 2.1 m/s^2), mean deceleration (4.1 ± 0.8 vs 2.7 ± 0.3 m/s^2), the rate-corrected preejection period (3.09 ± 0.75 vs 3.97 ± 0.22 $ms^{-1/2}$) and the preejection period to ejection time ratio (0.23 ± 0.07 vs 0.32 ± 0.01) were significantly different. No significant differences for Doppler parameters and pulmonary hemodynamics could be found between each of the 4 respiratory phases. Except for a higher maximal blood flow velocity, no significant difference in Doppler parameters was present in children with and without atrial septal defect.

Pearson correlations between Doppler parameters and PA pressure were generally low. The multiple regression equations relating PA hemodynamics with Doppler parameters were: PA systolic pressure = 2.70 · mean acceleration + 2.02 · maximal deceleration (R^2 0.40, SEE 11.0 mm Hg); PA mean pressure = 1.72 · mean acceleration + 1.06 · maximal deceleration (R^2 0.38, SEE 7.0 mm Hg); PA diastolic pressure = 23.1 − 44.5 · acceleration time/ejection time + 0.81 · maximal deceleration (R^2 0.32, SEE 4.8 mm Hg); driving force = 2.03 · mean acceleration − 6 (R^2 0.36, SEE 7.0 mm Hg); and PA vascular resistance index = 180.1 · mean acceleration (R^2 0.15, SEE 1.14 $U \cdot m^2$). Exclusion of the 3 patients with complete right bundle branch block did not improve correlation of Doppler indexes with PA hemodynamics. Multiple stepwise linear discriminant analysis resulted in the following function: 3.79 + 0.24 · maximal deceleration − 0.38 · mean acceleration + 0.58 · preejection period/($\sqrt{RR}$ interval), discriminating between presence (<0) or absence (>0) of pulmonary hypertension. Applying the discriminant function to our

patients resulted in a 91% accuracy (Table III). Sensitivity and specificity were 79% and 96%, respectively. The predictive value of a positive test was 92%, and that of a negative test 90%. Discriminative accuracy improved to 97% when comparing patients with PA mean pressure ≥30 mm Hg (n = 7) and <20 mm Hg (n = 28), with sensitivity of 100%, specificity of 96%, and a positive and negative predictive value of 88% and 100%. Classification of the 3 patients with right bundle branch block was correct.

DISCUSSION

This study investigates deceleration of PA blood flow velocity, because resistance to flow in pulmonary hypertension is changed during both flow acceleration and deceleration, and clear changes in deceleration have been reported in pulmonary hypertension.[13] Moreover, deceleration is also more likely to be dependent on pulmonary vascular resistance than on right ventricular function when compared with acceleration due to ventricular deactivation during the deceleration part of systole.

A decrease in pulmonary vascular compliance as a result of greater distending pressure and structural changes in the vascular wall, causing higher pulse-wave velocity[8,19,20] and earlier arrival of reflected waves from peripheral pulmonary vessels,[20,21] leads to increased flow velocity acceleration and earlier deceleration. This study comparing pulmonary blood flow velocity acceleration and deceleration with pulmonary hemodynamics (on a beat-to-beat basis) showed both increased acceleration and deceleration with elevated PA pressure. Maximal deceleration weakly correlated with PA pressures, but did not correlate with PA resistance or driving force. In pulmonary hypertension, mean and maximal deceleration were increased, the latter not reaching statistical significance. This study is one of the few to note that in addition to acceleration, deceleration is faster at higher PA pressures.[13] In pulmonary hypertension, deceleration time remained the same despite faster deceleration due to increased maximal velocity. The changes in deceleration by pulmonary hypertension would ideally make noninvasive PA pressure estimation possible. Because correlations between PA pressure and Doppler-derived deceleration parameters were too low, accurate prediction of PA pressures is not feasible using deceleration parameters alone.

The results of this study concerning the relation of acceleration with pulmonary hemodynamics are in concordance with published reports. In contrast to our hypothesis, acceleration phase–derived parameters were more closely related to PA pressures than those acquired during deceleration. However, the relation of acceleration to PA hemodynamics was also not strong enough for accurate noninvasive PA pressure estimation in this study. This weak relation could be the result of variability in Doppler parameter measurements or caused by mechanical ventilation. To diminish the effects of beat-to-beat variability, the median of 6 consecutive cardiac cycles was used in statistical analysis. Variability caused by mechanical ventilation may play a major role, but additional analysis taking respiratory phase into account did not improve correlations, and no significant differences between respiratory phases could be found for all Doppler parameters. This was probably due to the very small PA pressure changes during respiration and the small number of patients. Whether instantaneous changes in right ventricular loading conditions during positive pressure ventilation influence the relation between Doppler measurements and PA pressures is not clear. During inflation, right ventricular systolic pressure and volume increase and ejection fraction markedly decreases[22] causing acceleration to become less. By contrast, PA resistance elevation by inflation would shorten and increase acceleration by pulse wave reflection. In this study, small but insignificant elevations of PA pressure occurred during inflation, suggesting elevated resistance to flow during inspiration.

Determination of mean acceleration, maximal deceleration, and the rate-corrected preejection period allows accurate discrimination between presence and absence of pulmonary hypertension. Positive and negative predictive values were >90%. Because the discriminant function has been determined for this particular study population and accuracy would be lower in other populations, it should be used cautiously. Nevertheless, parameters concerning both acceleration, deceleration, and right ventricular function (preejection period) give important information on the presence of pulmonary hypertension.

The PA blood flow velocity waveform is determined not only by pulmonary hemodynamics, but also by right ventricular function.[7] The effect of right ventricular contractility on acceleration time can be offset by using the preejection period to acceleration time ratio in adults,[3] but not in infants.[10] That correction for heart rate of systolic time intervals did not result in better correlations with pulmonary hemodynamics is in agreement with our and other investigations,[6,10] although contrasting results have also been reported.[7,12] Acceleration time is shortened in severe tricuspid regurgitation due to diminished afterload.[23,24] Pulmonary regurgitation similarily can cause changes in Doppler-derived parameters by altering preload and stroke volume. Neither severe tricuspid regurgitation nor pulmonary regurgitation was present in our patients, but accuracy may have been influenced by moderate or even trivial right-sided regurgitation. Presence of right bundle branch block could lead to false-negative predictions for the presence of pulmonary hypertension,[7] but classification in 3 patients with this block was correct in this study. In atrial septal defect, normal acceleration times can be present despite the presence of elevated PA pressure and resistance.[8] Except for a higher maximal blood flow velocity, no significant difference in Doppler parameters, including acceleration

time, was present in children with and without atrial septal defect.

Another limitation in our study was the use of fluid-filled catheters, inevitably introducing artifacts in pressure measurements despite adequate frequency response. Digital signal filtering cannot entirely correct for these artifacts. Placement of the Doppler sample volume exactly in the middle of the main PA is difficult, but it is important because acceleration and deceleration differ near the vessel wall by its curvature. By using both the short-axis and long-axis view, error was minimized.

From this study investigating noninvasive determination of pulmonary hemodynamics in mechanically ventilated children with congenital heart disease, one can conclude that accurate prediction of PA pressure and resistance is not feasible by analysis of the pulmonary blood flow velocity waveform. An accurate prediction for the presence of pulmonary hypertension can, however, be made by measurement of acceleration as well as deceleration Doppler parameters.

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1. Kitabitake A, Inoue M, Asao M, Masuyama T, Tanouchi J, Morita T, Mishima M, Uematsu M, Shimazu T, Hori M, Abe H. Noninvasive evaluation of pulmonary hypertension by a pulsed Doppler technique. *Circulation* 1983;68: 302–309.
2. Martin-Duran R, Larman M, Trugeda A, Vazquez de Prada, Ruano J, Torres A, Figueroa A, Pajaron A, Nistal F. Comparison of Doppler-determined elevated pulmonary arterial pressure with pressure measured at cardiac catheterization. *Am J Cardiol* 1986;57:859–863.
3. Isobe M, Yazaki Y, Takaku F, Koizumi K, Hara K, Tsuneyoshi H, Yamaguchi T, Maechii K. Prediction of pulmonary arterial pressure in adults by pulsed Doppler echocardiography. *Am J Cardiol* 1986;57:316–321.
4. Stevenson JG. Comparison of several noninvasive methods for estimation of pulmonary artery pressure. *J Am Soc Echo* 1989;2:157–171.
5. Okamoto M, Miyatake K, Kinoshita N, Sakakibara H, Nimura Y. Analysis of blood flow in pulmonary hypertension with the pulsed Doppler flowmeter combined with cross sectional echocardiography. *Br Heart J* 1984;51:407–415.
6. Kosturakis D, Goldberg SJ, Allen HD, Loeber C. Doppler echocardiographic prediction of pulmonary arterial hypertension in congenital heart disease. *Am J Cardiol* 1984;53:1110–1115.
7. Serwer GA, Cougle AG, Eckerd JM, Armstrong BE. Factors affecting use of the Doppler-determined time from flow onset to maximal pulmonary artery velocity for measurement of pulmonary artery pressure in children. *Am J Cardiol* 1986;58:352–356.
8. Matsuda M, Sekiguchi T, Sugishita Y, Kuwako K, Iida K, Ito I. Reliability of non-invasive estimates of pulmonary hypertension by pulsed Doppler echocardiography. *Br Heart J* 1986;56:158–164.
9. Dabestani A, Mahan G, Gardin JM, Takenaka K, Burn C, Allfie A, Henry WL. Evaluation of pulmonary artery pressure and resistance by pulsed Doppler echocardiography. *Am J Cardiol* 1987;59:662–668.
10. Salzer-Muhar U, Schlemmer M, Proll E, Salzer HR, Wimmer M. Doppler echocardiographic assessment of pulmonary arterial pressure in children with congenital heart disease. *Wien Klin Wochenschr* 1986;98:744–747.
11. Graettinger WF, Greene ER, Voyles WF. Doppler predictions of pulmonary artery pressure, flow, and resistance in adults. *Am Heart J* 1987;113:1426–1437.
12. Akiba T, Yoshikawa M, Otaki S, Kobayashi Y, Nakasato M, Suzuki H, Sato T. Prediction of peak pulmonary artery pressure by continuous-wave Doppler echocardiography in infants and children. *Pediatr Cardiol* 1988;9:225–229.
13. Ritter SB, Cooper RS, Golinko RJ. Noninvasive assessment of pulmonary hypertension and pulmonary vascular reactivity in congenital heart disease: pulsed Doppler application. *J Cardiovasc Ultrason* 1986;5:213–221.
14. Panidis IP, Ross J, Mintz GS. Effect of sampling site on assessment of pulmonary artery blood flow by Doppler echocardiography. *Am J Cardiol* 1986;58:1145–1147.
15. Shaffer EM, Snider R, Serwer GA, Peters J, Reynolds PA. Effect of sampling site on Doppler-derived right ventricular systolic time intervals. *Am J Cardiol* 1990;65:950–952.
16. Morady F, Brundage BH, Gelberg HJ. Rapid method for determination of shunt ratio using a thermodilution technique. *Am Heart J* 1983;106:369–373.
17. Grossman W. Shunt detection and measurement. In: Grossman W, ed. Cardiac Catheterization and Angiography. Philadelphia: Lea & Febiger, 1986:155–169.
18. Grossman W. Clinical measurement of vascular resistance and assessment of vasodilator drugs. In: Grossman W, ed. Cardiac Catheterization and Angiography. Philadelphia: Lea & Febiger, 1986:135–142.
19. Reuben SR. Compliance of the human pulmonary arterial system in disease. *Circ Res* 1971;29:40–50.
20. Milnor WR, Conti CR, Lewis KB, O'Rourke MF. Pulmonary arterial pulse wave velocity and impedance in man. *Circ Res* 1969;25:637–649.
21. Matsuda M, Sugishita Y, Yamaguchi T, Tamura T, Ito I. Pulmonary flow velocity patterns in patients with pulmonary hypertension: a study using pulsed Doppler echocardiography combined with two-dimensional echocardiography. *J Cardiogr* 1981;11:603–614.
22. Jardin F, Delorme G, Hardy A, Auvert B, Beauchet A, Bourdarias J. Reevaluation of hemodynamic consequences of positive pressure ventilation: emphasis on cyclic right ventricular afterloading by mechanical lung inflation. *Anesthesiology* 1990;72:966–970.
23. Hatle L, Angelsen B. Doppler ultrasound in cardiology. In: Hatle L, Angelsen B, eds. Physical Principles and Clinical Applications. 2nd ed. Philadelphia: Lea & Febiger, 1985:261.
24. Morrisson DA, Ovitt T, Hammermeister KE. Functional tricuspid regurgitation and right ventricular dysfunction in pulmonary hypertension. *Am J Cardiol* 1988;62:108–112.